repair; A4, 14 patients who had single ventricle and were treated with Fontan type operation. Group B was subdivided into 3 groups; B1, 6 patients with C-CHD palliated by classic Glenn procedure; B2, 6 patients with C-CHD palliated by shunt procedure; B3, 9 healthy controls. There was no significant difference in age among subgroups of A and among subgroups of B. Blood samples were obtained from upper arm veins and serum VEGF was determined by ELISA method. We determined correlation between patient's arterial saturation and serum VEGF in combined group of A1, A2, B1, B2 and B3 and compared level of serum VEGF among groups. Data were expressed as median and inter-quartile range in parenthesis. RESULTS: Serum VEGF was significantly negatively correlated with arterial saturation (r=.48, p<.001). Serum VEGF in A1 was significantly higher than those in A2 and A3 [373(269) pg/ml in A1 vs. 196(121) pg/ml in A2 and 186(87) pg/ml in A3, respectively], but not higher than VEGF in A4 [257(293) pg/ml]. Although it did not reach significant level, serum VEGF in A4 tended to be higher than A3 (p=.07). Serum VEGF in B1 and B2 was significantly higher than in B3 [269(379) pg/ml in B1 and 657(382) pg/ml in B2 vs. 225(105) pg/ml in B3, respectively]. There was no significant difference in serum VEGF level between B1 and B2. CONCLUSIONS: Patients with C-CHD seem to have increased serum VEGF in parallel with the degree of cyanosis. Patients with C-CHD palliated by classic Glenn procedure do have increased serum VEGF, but not more than those palliated by shunt procedure. With bi-ventricular repair, cyanosis disappears and serum VEGF may be normalized. In contrast, with Fontan type operation, cyanosis disappears but serum VEGF may not be normalized

1:48 p.m.

1121MP-130 Leukocyte Adhesion Factor Mac-1 and Migration Inhibitory Factor-Related Protein (MRP) on Granulocyte Plays the Essential Role for Causing Vasculitis in Kawasaki Disease and the Gamma Globulin Therapy Inhibit Leukocyte-Endothelial Cell Adhesion

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(Background) We have revealed massive expression of Ca binding protein migration inhibitory factor-related protein(MRP) on circulating granulocyte in acute phase of Kawasaki disease. Newton et. al. (J Immunol 1998 160:1427) reported MRP reinforce the ability of adhesion molecule Mac-1, which suggest the relationship between MRP/Mac-1 and vasculitis. We quantified leukocytes Mac-1 expression in Kawasaki disease, and evaluated the adhesion ability between cultured human coronary artery endothelial cell and Kawasaki disease patient's peripheral leukocyte. Furthermore, we evaluate the leukocyte-endothelial cell adhesive ability using the patients' plasma, before and after gamma globulin therapy, to see the gamma globulin effect for this system. **(Materials and Methods)** mRNA was extracted from the Kawasaki disease patients' leukocyte (n=21) and was converted to cDNA by RT-PCR, and Mac-1 expression was evaluated by quantitative PCR (Applied Biosystems; GeneAmp 5700). Patients' leukocyte, labeled with BCECF-AM, exposed to cultured human coronary artery endothelial cell, and leukocyte adhesion assay was performed. Leukocyte adhesion assay was also carried out using the patients' plasma, pre/post gamma globulin therapy. **(Result)** Mac-1 expression was a peak on acute phase of Kawasaki disease and significantly decreased after 1 month of onset. The patients' leukocyte adhesion ability to endothelial cell was significantly increased, which was significantly inhibited by addition of anti-Mac-1 antibody. And the patients' plasma before gamma globulin therapy significantly increased leukocyte-endothelial cell adhesion, which was abolished by the plasma of post gamma globulin therapy. We postulated Mac-1 play the key role for leukocyte invasion into endothelium, which is the initial step for causing vasculitis. And the gamma globulin therapy is effective through inhibiting leukocyte-endothelial cell adhesion.

POSTER SESSION

1142 Pediatric Electrophysiology and Intervention

Monday, March 18, 2002, 3:00 p.m.-5:00 p.m.
Georgia World Congress Center, Hall G
Presentation Hour: 4:00 p.m.-5:00 p.m.

1142-97 Termination of Intraatrial Reentrant Tachycardia Using Implanted Pacemakers in Patients Following Atrial Switch Repair of Transposition of the Great Arteries

Gerald A. Serwer, David J. Bradley, Peter S. Fischbach, Kristen A. George, Sarah S. Leroy, Macdonald Dick, II, *University of Michigan Congenital Heart Center, Ann Arbor, Michigan.*

Background: Intra-atrial reentrant tachycardia (IART) has been associated with patients (pts) following the atrial switch operation (AtrS) for d-transposition of the great arteries who have sick sinus syndrome (SSS). Anti-tachycardic pacing (ATP) has been used to convert IART in these pts but its efficacy using implanted pacemakers and safety have been questioned.
Methods: Clinical records of pts post AtrS who had undergone atrial or dual chamber pacemaker placement for SSS were retrospectively reviewed. Implant details, number and results of ATP attempts, medications, and outcomes were recorded.
Results: Of the 29 pts identified, atrial electrode placement was endocardial in 25 and epicardial in 4 and was guided only by pacing threshold data with no attempt to induce and convert IART at implant. 21 (72%) had 1 or more episodes of IART and all were taking digoxin, 5 on beta-blockers, and 2 on amiodarone. 4 of 21 had IART prior to pacemaker implant without recurrence post implant. 17 underwent ATP for conversion of 1 to 30 episodes per patient (94 total episodes, median 3/patient). 7 pts experiencing 47 episodes had automatic anti-tachycardic pacemakers. ATP therapy consisted of 15 to 40 beats at a cycle length of 50% to 80% of the IART cycle length (median 67%, minimum 150 ms) delivered at an amplitude and pulse width twice the bradycardia pacing settings. ATP cycle length began at 80% of the IART cycle length and was decreased until conversion. All episodes were converted but many required multiple attempts. 8 ATP attempts increased the IART rate but not the ventricular rate. 4 ATP attempts in 2 pts caused atrial fibrillation that reverted spontaneously to sinus rhythm. All pts were hemodynamically stable in IART before and during ATP.
Conclusion: Bradycardia pacing can control IART in some pts. ATP by implanted pacemakers is safe and effective in converting IART in pts post AtrS procedure who are not severely hemodynamically compromised by IART avoiding the need for DC cardioversion. IART induction is not required at pacemaker implant. Pacemakers with atrial overdrive capability should be considered in pts post AtrS with SSS as the incidence of IART is high and ATP is highly effective and safe.

1142-98 Comparison of Adenosine-Sensitive and Adenosine-Resistant Accessory Pathways in Children

David J. Bradley, Loren M. Madden, Christopher B. Stefanelli, Elizabeth V. Saarel, Jeremy D. Asnes, Gerald A. Serwer, Peter S. Fischbach, Macdonald Dick, II, *University of Michigan Congenital Heart Center, Ann Arbor, Michigan.*

Background: Adenosine (Adn) is routinely used during electrophysiolgic study (EPS) of patients with supraventricular tachycardia (SVT) to assess presence of accessory atrioventricular pathways (AP) and verify success of radiofrequency ablation (RFA). Adenosine blocks conduction in the atrioventricular node, but generally not in APs. We investigated the incidence and properties of Adn-sensitive APs.
Methods: The electrophysiologic patient database at the University of Michigan Congenital Heart Center was queried for all patients with accessory pathways and SVT undergoing EPS between 4-1995 and 4-2001. Clinical records and EPS tracings were reviewed. Patients received Adn during ventricular pacing at a standard intravenous dose of 200mcg/kg, with a maximum dose of 12mg was used. All patients were under 21 years of age (median 11.6 years).
Results: A total of 151 patients with APs and SVT were identified; Adn was used in 132 patients (137 APs). Of these, 20 APs (15%) blocked with adenosine. RFA was performed on 85% (117/137) APs, and was successful in 94% (15/16) Adn-sensitive, v 92% (93/101) Adn-insensitive APs (p=ns). Adn-sensitive and -insensitive APs did not differ significantly with regard to patient age (10.6±5.8 v 11.4±4.7 years), gender (47% v 55% boys), or SVT cycle length (314±39 v 315±46ms), procedure times, total RF applications or rate of ablation success. Adn-sensitive and insensitive pathways did not segregate by septal v free-wall (35% v 28% septal, p=0.16), or left v right sided (63% v 60% left, p=0.36) locations. In contrast, Adn-sensitive APs were less likely to demonstrate pre-excitation in sinus rhythm (37% v 61%, p=0.04), and had significantly longer antegrade effective refractory periods (ERP) when pre-excitation was present (340±42 v 278±58ms; p=0.05). Retrograde ERP also trended longer in Adn-sensitive pathways (270±57 v 259±43) though this difference was not statistically significant (p=0.10).
Conclusions: As Adn-sensitive APs occur frequently in pediatric patients, Adn is not an absolute test of AP presence. Adn-sensitive APs are significantly less likely than Adn-resistant APs to cause pre-excitation, and have longer antegrade ERPs when they do.

1142-99 Biologic Response to the HELEX™ Septal Occluder Implantation in the Canine Heart

Geoffrey K. Lane, Nicholas P. Macri, John F. Rhodes, C.Igor Mesia, Lourdes R. Prieto, Paul L. Sawyer, Melanie M. Manning, Dena J. Anderson, Evan M. Zahn, Larry A. Latson, *The Cleveland Clinic Foundation, Cleveland, Ohio, W. L Gore & Associates Inc, Flagstaff, Arizona.*

Background: The HELEX™ device has recently been used in humans for transcatheter secundum atrial septal defect (ASD) closure. We report the data on the biologic response to implantation of this device in the canine heart. **Methods:** Histologic data from 23 animals enrolled in non-randomized prospective studies conducted at W.L. Gore & Associates Inc and The Cleveland Clinic Foundation were analyzed. Animals had either a surgically created or a percutaneously created ASD and immediate implantation of a HELEX™ device. Animals were sacrificed at intervals from 2 days to 1 year. Devices were examined grossly, with multiple sections of the device and adjacent atrial septum assessed for fibrous connective tissue (FCT) and endothelial-like cell coverage. Distal organs were examined for the presence of thrombo-emboli in animals with implanted devices ≥ 30 days. **Results:** FCT completely covered the left atrial component in 12/13 devices, which had been implanted ≥ 30 days. One device at 6 months post implantation had complete coverage of the right atrial side with extensive coverage of the left atrial side. Endothelial-like cell coverage was multifocal or greater in 10/13 devices implanted ≥ 30 days. One device examined by scanning electron microscopy and immunohistochemistry (for factor VIII) confirmed that the endothelial-like cells seen with light microscopy were true endothelial cells. There was a trend for greater FCT and endothelial cell coverage of devices with time. There was no evidence of distal organ thromboembolism in any animal examined. **Conclusions:** The biologic response to implantation of the HELEX™ device appears to be progressive with an initial fibrous connective tissue coverage followed by endothelialization. There was complete coverage of the left atrial side of the device with fibrous connective tissue by 30 days post implantation in almost all cases with no evidence of distal thromboembolism. These data support the notion that the HELEX™ device is biocompatible. Further research into the process of endothelialization of this and other transcatheter devices is needed.